

Type	L #	Hits	Search Text	DBS	Time Stamp	Cor rel ati on	Er ro r m en ti on
1	BRS	L1	2365	septicemia	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:45	0
2	BRS	L2	2602	lbp or (liposaccharide adj binding adj protein)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:45	0
3	BRS	L3	0	1 same 2	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:46	0
4	BRS	L4	221	bactericidal\$1permeability adj increasing adj protein	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:46	0
5	BRS	L5	215	bactericidal/permeability adj increasing adj protein	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:46	0
6	BRS	L6	221	4 or 5	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:47	0
7	BRS	L7	37	limulus adj anti-LPS adj factor	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:47	0
8	BRS	L8	53	limulus adj anti-LPS adj factor or lalf	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/0 8 11:47	0

Type	L #	Hits	Search Text	DBs	Time Stamp	Cor nm en ts	Ex ro Er De fi rs ni ti on
9	BRS	L9	53	7 or 8	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/08 11:48	0
10	BRS	L11	0	2 same (6 or 9) same hybrid	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/08 11:48	0
11	BRS	L10	19	2 same (6 or 9)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/08 11:54	0
12	BRS	L12	1	schumann adj ralf adj reiner.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/08 11:55	0
13	BRS	L13	2	schumann adj ralf.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/08 11:56	0
14	BRS	L14	3	lamping adj norbert.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/04/08 11:56	0

FILE 'MEDLINE' ENTERED AT 12:01:40 08 APR 2003

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=> s septicemia  
L1 55506 SEPTICEMIA

=> s lipopolysaccharide binding protein  
L2 2979 LIPOPOLYSACCHARIDE BINDING PROTEIN

=> s l1 (p) l2  
L3 19 L1 (P) L2

=> duplicate remove l3  
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE'  
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n  
PROCESSING COMPLETED FOR L3  
L4 17 DUPLICATE REMOVE L3 (2 DUPLICATES REMOVED)

=> d 14 1-17 ibib abs

L4 ANSWER 1 OF 17 MEDLINE DUPLICATE 1  
ACCESSION NUMBER: 2002634766 MEDLINE  
DOCUMENT NUMBER: 22280781 PubMed ID: 12394943  
TITLE: Characterization of a myocardial depressant factor in meningococcal septicemia.  
COMMENT: Comment in: Crit Care Med. 2002 Oct;30(10):2379-80  
AUTHOR: Pathan Nazima; Sandiford Colin; Harding Sian E; Levin Michael  
CORPORATE SOURCE: Department of Pediatrics, Imperial College of Science, Technology and Medicine, London, UK.  
SOURCE: CRITICAL CARE MEDICINE, (2002 Oct) 30 (10) 2191-8.  
Journal code: 0355501. ISSN: 0090-3493.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200211  
ENTRY DATE: Entered STN: 20021024  
Last Updated on STN: 20021213  
Entered Medline: 20021107

AB OBJECTIVE: Identification and characterization of myocardial depressant factors present in meningococcal \*\*\*septicemia\*\*\* . DESIGN: Laboratory investigation of myocardial depression that used isolated cardiac myocytes as an model of cardiac contractile function. SETTING: University hospital and laboratories. PATIENTS: Children with severe meningococcal septic shock requiring intensive care. ANIMALS: Myocytes obtained from adult male Sprague-Dawley rats. INTERVENTIONS: Serum samples obtained from the acute phase of sepsis were evaluated for the presence of myocardial depressant activity. Further characterization of the myocardial depressant factor was undertaken by using cell culture supernatants from whole blood and peripheral blood mononuclear cells that had been exposed to heat-killed meningococci. MEASUREMENTS AND MAIN RESULTS: Myocardial depressant activity was measured by using isolated rat left-ventricular myocytes. Changes in amplitude of contraction and in the speed of contraction and relaxation were determined after cells were exposed to various stimuli. Serum from patients with meningococcal disease had myocardial depressant activity. This activity was also present in whole blood and peripheral blood mononuclear cells exposed to meningococci. Myocardial depressant

activity was found to be heat stable, proteinaceous, and of a molecular weight range of 10-25 kDa. The activity did not elevate concentrations of cyclic guanylic acid. \*\*\*Lipopolysaccharide\*\*\* - \*\*\*binding\*\*\*

\*\*\*protein\*\*\* augmented the release of myocardial depressant factor by peripheral blood mononuclear cells exposed to meningococci. CONCLUSIONS: Myocardial depression in meningococcal sepsis is mediated in part by circulating myocardial depressant factors. Myocardial depressant factors are also released when whole blood or peripheral blood mononuclear cells of healthy donors are exposed to heat-killed meningococci. Release of the factors appears to be mediated through endotoxin-induced activation of peripheral blood mononuclear cells, since \*\*\*lipopolysaccharide\*\*\* - \*\*\*binding\*\*\* \*\*\*protein\*\*\* augments release in a dose-responsive manner. Partial physicochemical characterization of the factors has been achieved.

L4 ANSWER 2 OF 17 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2001044742 EMBASE

TITLE: Endotoxin and adjunctive therapies in gram-negative sepsis.

AUTHOR: Romano M.J.

CORPORATE SOURCE: Dr. M.J. Romano, Clinical Pediatrics, Texas Tech Univ. Health Sci. Center, 3601 4th St, Lubbock, TX 79430, United States

SOURCE: Seminars in Pediatric Infectious Diseases, (2001) 12/1 (17-23).

Refs: 100

ISSN: 1045-1870 CODEN: SPIDFJ

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 007 Pediatrics and Pediatric Surgery  
026 Immunology, Serology and Transplantation  
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Gram-negative infections remain a major cause of morbidity and mortality. The death rate from \*\*\*septicemia\*\*\* is increasing and now surpasses acquired immunodeficiency syndrome as a cause of death. Initial attempts at adjunctive therapy using monoclonal antibodies directed against the lipid A core of endotoxin (HA-1A, E5) have not provided consistent benefit in large-scale trials. The only other agent targeting endotoxin is bactericidal permeability increasing protein, for which a phase III trial in children with meningococcemia has been completed. Other strategies being developed include modifying the effect of mediators of endotoxin toxicity, including CD14, soluble CD14, and \*\*\*lipopolysaccharide\*\*\* - \*\*\*binding\*\*\* \*\*\*protein\*\*\* ; detoxifying endotoxin by binding with polymyxin B; and implementing antagonists of endotoxin or compounds that inhibit the biosynthesis of endotoxin. Copyright .COPYRGT. 2001 by W.B. Saunders Company.

L4 ANSWER 3 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:59315 CAPLUS

DOCUMENT NUMBER: 128:106451

TITLE: Nonoral preparations containing phospholipids, and prevention and treatment of septicemia with them

INVENTOR(S): Kanagi, Yoshiaki; Ono, Yoichi; Mori, Masato; Akiyama, Tadashi

PATENT ASSIGNEE(S): Nippon Seiyaku K. K., Japan

SOURCE: Jpn. Kokai Tokkyo Koho, 8 pp.  
CODEN: JKXXAF

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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JP 10017476	A2	19980120	JP 1996-188173	19960628
PRIORITY APPLN. INFO.:			JP 1996-188173	19960628

AB The preps. for \*\*\*septicemia\*\*\* contain phospholipids suspended therein, and the phospholipids mobilize lipoproteins when i.v. administered, thus inactivating endotoxins and/or endotoxin- \*\*\*lipopolysaccharide\*\*\* - \*\*\*binding\*\*\* \*\*\*protein\*\*\* complexes. \*\*\*Septicemia\*\*\* is prevented or treated by i.v. administration of the

preps. Thus, yolk lecithin was homogenized with an aq. glycerin soln. to give a suspension. Escherichia coli LPS was injected to rats [REDACTED] 4th day of 5-day consecutive i.v. administration of the suspension to cause no death of the animals.

L4 ANSWER 4 OF 17 CAPLUS COPYRIGHT 2003 ACS  
ACCESSION NUMBER: 1998:398796 CAPLUS  
DOCUMENT NUMBER: 129:53159  
TITLE: Flow cytometric analysis of immunoparalysis  
AUTHOR(S): Nebe, Carl Thomas  
CORPORATE SOURCE: Fak. Klin. Med., Inst. Klin. Chem., Univ. Heidelberg,  
Mannheim, D-68135, Germany  
SOURCE: Clinical Laboratory (Heidelberg) (1998), 44(6),  
441-446  
CODEN: CLLAfp  
PUBLISHER: Clin Lab Publications  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB To diagnose immunoparalysis and prognosis of \*\*\*septicemia\*\*\*, an assay for the detn. of the cell surface d. of HLA-DR expression on monocytes in peripheral blood was standardized and validated. Monocytes were identified by their receptor for the \*\*\*lipopolysaccharide\*\*\* - \*\*\*binding\*\*\* \*\*\*protein\*\*\* CD14, while the expression d. of antigen-presenting mols. on their cell surface was registered via immunofluorescence. Diminished expression of antigen-presenting mols. (major histocompatibility complex class II, human leukocyte antigen-DR) was assocd. with high risk of sepsis and lethal outcome. Fluorescence intensity above 250 channels was normal and values >50 correlated with a poor prognosis.

L4 ANSWER 5 OF 17 CAPLUS COPYRIGHT 2003 ACS  
ACCESSION NUMBER: 1995:982430 CAPLUS  
DOCUMENT NUMBER: 124:764  
TITLE: Recombinant preparation of polypeptides of human lipopolysaccharide binding protein and use of the polypeptides for sepsis treatment  
INVENTOR(S): Han, Jiahuai; Ulevitch, Richard J.; Tobias, Peter S.  
PATENT ASSIGNEE(S): Scripps Research Institute, USA  
SOURCE: PCT Int. Appl., 63 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9525117	A1	19950921	WO 1995-US3384	19950315
W: AU, CA, FI, JP, NO				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5837810	A	19981117	US 1994-215089	19940315
AU 9521868	A1	19951003	AU 1995-21868	19950315
PRIORITY APPLN. INFO.:			US 1994-215089	19940315
			WO 1995-US3384	19950315

AB A polypeptide fragment of lipopolysaccharide (LPS)-binding protein (LBP) that inhibit the binding of LPS released by gram-neg. bacteria into the CD14 receptor is provided. A method of ameliorating symptoms of sepsis in a subject by administration of an LBP polypeptide of the invention, or administration of antibody to LBP polypeptide, or anti-idiotype antibody is also disclosed.

L4 ANSWER 6 OF 17 CAPLUS COPYRIGHT 2003 ACS  
ACCESSION NUMBER: 1995:741240 CAPLUS  
DOCUMENT NUMBER: 123:141733  
TITLE: Monoclonal antibodies immunoreactive with lipopolysaccharide binding protein (LBP) and methods of their use  
INVENTOR(S): Kirkland, Theo; Tobias, Peter; Ulevitch, Richard; Moriarty, Ann; Leturcq, Didier  
PATENT ASSIGNEE(S): Scripps Research Institute, USA  
SOURCE: PCT Int. Appl., 76 pp.  
CODEN: PIXXD2

DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9514039	A1	19950526	WO 1994-US13199	19941115
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9512095	A1	19950606	AU 1995-12095	19941115
US 5753504	A	19980519	US 1996-679944	19960715
US 6232080	B1	20010515	US 1998-81350	19980519
US 2001029292	A1	20011011	US 2001-858279	20010515
PRIORITY APPLN. INFO.:			US 1993-153364	A 19931116
			WO 1994-US13199	W 19941115
			US 1996-679944	A3 19960715
			US 1998-81350	A3 19980519

AB The present invention concerns a method of treating LBP-mediated LPS-induced myeloid cell activation comprising administering a therapeutically effective amt. of an anti-LBP monoclonal antibody mol. A therapeutic compn. comprising anti-LBP antibody mols. in a pharmaceutically acceptable excipient is also contemplated. The LBP-mediated LPS-induced myeloid cell activation may relate to sepsis or gram-neg. bacterial infection. The method may also include antibiotic treatment. In example, anti-LBP monoclonal antibodies were prep., characterized, and used in inhibiting LBP-mediated cell activation. The inhibition included LBP-mediated binding of LPS to CD14, secretion of tumor necrosis factor from myeloid cells, etc.

L4 ANSWER 7 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1995:967850 CAPLUS  
DOCUMENT NUMBER: 124:75647  
TITLE: Activity of lipopolysaccharide-binding protein-bactericidal/permeability-increasing protein fusion peptide in an experimental model of Pseudomonas sepsis  
AUTHOR(S): Opal, Steven M.; Palardy, John E.; Jhung, Jhung W.; Donsky, Curtis; Romulo, Rodrigo L. C.; Parejo, Nicholas; Marra, Marian N.  
CORPORATE SOURCE: Memorial Hosp. Rhode Island, Brown Univ. Sch. Med., Providence, RI, USA  
SOURCE: Antimicrobial Agents and Chemotherapy (1995), 39(12), 2813-15  
PUBLISHER: CODEN: AMACQ; ISSN: 0066-4804  
DOCUMENT TYPE: American Society for Microbiology  
LANGUAGE: Journal English

AB A chimeric protein consisting of the N-terminal domain of lipopolysaccharide-binding protein and the C-terminal domain of bactericidal/permeability-increasing protein demonstrated a dose-dependent survival benefit ( $P = 0.001$ ) and reduced endotoxin levels ( $P < 0.01$ ) in neutropenic rats with *Pseudomonas aeruginosa* sepsis. This lipopolysaccharide-binding protein-bactericidal/permeability-increasing peptide has favorable pharmacokinetics and antiendotoxin properties which may be of value for human sepsis.

L4 ANSWER 8 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:657645 CAPLUS  
DOCUMENT NUMBER: 125:324844  
TITLE: Contribution of lipopolysaccharide binding protein (LBP) in endotoxemic shock in mice  
AUTHOR(S): Heumann, Didier; Gallay, Philippe; Le Roy, Didier; Glauser, Michel Pierre  
CORPORATE SOURCE: Division Infectious Diseases, Department Medicine, Lausanne, CHUV-1011, Switz.  
SOURCE: Progress in Clinical and Biological Research (1995), 392(Bacterial Endotoxins), 465-471  
PUBLISHER: CODEN: PCBRD2; ISSN: 0361-7742  
DOCUMENT TYPE: Wiley-Liss  
LANGUAGE: Journal English

AB The authors confirmed *in vivo* the role of LBP suggested by *in vitro* studies. In *vivo*, anti-LBP IgG afforded protection against lethal endotoxemia by one of 2 mechanisms: (1) LBP blockade by pretreatment with anti-LBP IgG allowed protection against a low lipopolysaccharide (LPS) challenge (100 ng); (2) anti-LBP treatment given simultaneously to LPS challenge protected mice independently from the dose used for challenge. Thus, anti-LBP could be a candidate for therapeutic strategies in endotoxemic shock, esp. when considering the fact that LBP is known to bind to a wide range of LPS and to lipid A.

L4 ANSWER 9 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1995:502408 CAPLUS  
DOCUMENT NUMBER: 122:263275  
TITLE: Induction of lipopolysaccharide-binding protein gene expression in cultured rat pulmonary artery smooth muscle cells by interleukin 1.beta.  
AUTHOR(S): Wong, Hector R.; Pitt, Bruce R.; Su, Grace L.; Rosignol, Daniel P.; Steve, A. Regina; Billiar, Timothy R.; Wang, Stewart C.  
CORPORATE SOURCE: Dep. of Anesthesiology/Critical Care Medicine, Univ. of Pittsburgh School of Medicine, Pittsburgh, PA, USA  
SOURCE: American Journal of Respiratory Cell and Molecular Biology (1995), 12(4), 449-54  
CODEN: AJRBEL; ISSN: 1044-1549  
PUBLISHER: American Lung Association  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Lipopolysaccharide (LPS)-binding protein (LBP) binds with high affinity to LPS, and the LBP-LPS complex enhances cellular inflammatory responses to LPS. Although it is present in normal serum, LBP is also induced as part of the acute phase response. Synthesis of LBP is thought to be limited to the liver, but we have recently reported significant extrahepatic (including pulmonary) LBP mRNA expression in *in vivo* rat models of sepsis and inflammation. In the present study, we tested the hypothesis that a cellular source of pulmonary LBP in the rat may be vascular smooth muscle, by exposing cultured rat pulmonary artery smooth muscle cells (RPASMC) to cytokines and LPS. Treatment of RPASMC for 4 and 24 h with a combination of tumor necrosis factor .alpha., interleukin 1.beta. (IL-1.beta.), interferon .gamma., and LPS resulted in significant LBP mRNA expression. Of this mixt., IL-1.beta. alone was sufficient to induce LBP mRNA expression in both a time- and dose-dependent manner. The effects of IL-1.beta. on LBP mRNA expression were significantly antagonized by IL-1 receptor antagonist protein. Furthermore, supernatants from RPASMC treated with IL-1.beta. enhanced the binding of [<sup>125</sup>I]ASD-LPS by the macrophage cell line RAW 264.7, indicative of LBP bioactivity. We conclude that pulmonary artery smooth muscle cells stimulated with IL-1.beta. produce a transcript for LBP or a homologous product *in vitro*. Local prodn. of LBP could play an important role in the pulmonary response to inflammation and sepsis.

L4 ANSWER 10 OF 17 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1996:45276 BIOSIS  
DOCUMENT NUMBER: PREV199698617411  
TITLE: LPS induces interleukin (IL)-1 converting enzyme (ICE) in monocytic and endothelial cells, as assessed by a novel ice bioassay using an artificial fluorogenic substrate.  
AUTHOR(S): Pfeil, D.; Belka, C.; Brach, M. A.; Kirschning, C.; Lamping, N.; Reuter, D.; Herrmann, F.; Schumann, R. R.  
CORPORATE SOURCE: Max-Delbrueck-Centrum Molekulare Med. und Abteilung Med. Onkol. Angewandte Molekularbiol., Humboldt-Univ. zu Berlin, Virchow-Klinikum, Robert-Roessle-Klin., D-13122 Berlin Germany  
SOURCE: Onkologie, (1995) Vol. 18, No. SUPPL. 2, pp. 125.  
Meeting Info.: Annual Congress of the German and Austrian Societies for Hematology and Oncology Hamburg, Germany October 8-11, 1995  
ISSN: 0378-584X.  
DOCUMENT TYPE: Conference  
LANGUAGE: English

L4 ANSWER 11 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:676191 CAPLUS

DOCUMENT NUMBER: 121:276191  
TITLE: Use of bactericidal/permeability-increasing protein (BPI) and lipopolysaccharide-binding protein (LBP) levels and ratios thereof in diagnosis of inflammatory disorder  
INVENTOR(S): Scott, Randal W.; Fisher, Charles J.; Marra, Marian N.; Opal, Steven M.  
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., USA  
SOURCE: PCT Int. Appl., 48 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9421280	A1	19940929	WO 1994-US3086	19940321
W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KG, KP, KR, KZ, LK, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9465227	A1	19941011	AU 1994-65227	19940321
PRIORITY APPLN. INFO.: US 1993-34294 19930322				
WO 1994-US3086 19940321				

AB Methods are provided for diagnosing and predicting the outcome of an inflammatory disorder in a subject, which comprise obtaining a bodily fluid sample from the patient, quant. detg. the amt. of BPI in the sample, comparing the amt. of BPI present in the sample with levels of BPI in samples obtained from normal subjects and with levels of BPI in samples obtained from individuals suffering from an inflammatory disorder in order to diagnose and predict the outcome of an inflammatory disorder in the subject. Alternatively, the amt. of BPI and LBP is detd. in the sample, the ratio of BPI to LBP is calcd., and the ratio is compared with those obtained from normal subjects and with those from individuals suffering from an inflammatory disorder in order to diagnose and predict the outcome of the inflammatory disease in the subject. The amts. are detd. by ELISA. BPI levels are elevated in vivo in response to endotoxin challenge and as a result of inflammatory disease. The elevated levels include free BPI and cell surface-bound BPI. BPI could be detected as early as 30 min following LPS treatment, and release was maximal at 2 h.

L4 ANSWER 12 OF 17 CAPLUS COPYRIGHT 2003 ACS  
ACCESSION NUMBER: 1994:555240 CAPLUS  
DOCUMENT NUMBER: 121:155240  
TITLE: Mode of action of anti-lipopolysaccharide-binding protein antibodies for prevention of endotoxemic shock in mice  
AUTHOR(S): Gallay, P.; Heumann, D.; Le Roy, D.; Barras, C.; Glauser, M. P.  
CORPORATE SOURCE: Dep. Internal Med., Div. Infectious Diseases, Lausanne, CHUV-1011, Switz.  
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1994), 91(17), 7922-6  
CODEN: PNASA6; ISSN: 0027-8424  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Lipopolysaccharide (LPS)-binding protein (LBP) has been shown to regulate the response of monocytes to LPS in vitro. In a previous study, polyclonal anti-LBP IgGs were found to protect D-galactosamine-sensitized mice against a lethal endotoxemic shock induced by a low challenge of LPS or lipid A when administered simultaneously with endotoxin. In the present study the authors investigated the mode of action of these anti-LBP IgGs. In vitro, the authors demonstrated that they interfere with LPS binding to monocytes or polymorphonuclear cells in different ways: by the mere prevention of binding of LPS to LBP thus preventing the binding of LPS to CD14, or by reacting with LPS-LBP complexes thus mediating their binding to complement or Fc receptors on monocytes and on polymorphonuclear cells. In vivo, the authors demonstrated that anti-LBP IgGs afforded protection against lethal endotoxemic shock by one of two mechanisms. First, LBP blockade by pretreatment with anti-LBP IgG allowed

protection against a low dose of LPS (100 ng). This protection occurred despite LPS levels in blood similar to those in control mice b. in the absence of detectable tumor necrosis factor (TNF). This demonstrated that anti-LBP IgG could block the LBP-mediated TNF release upon LPS challenge. In contrast, anti-LBP IgG did not afford protection in mice not sensitized with D-galactosamine and challenged with high-dose LPS (1 mg), confirming that LPS at high concn. could stimulate cells independently of the LBP pathway. Second, anti-LBP treatment administered simultaneously with LPS challenge protected mice against both low and high doses of LPS. Unlike after pretreatment with anti-LBP IgG, this protection was accompanied by a decrease of circulating LPS, suggesting that anti-LBP IgG in these conditions facilitated clearance of LPS probably by clearing LPS-LBP complexes. These data and the fact that LBP binds to all LPS through lipid A suggest that antibody directed to LBP could be a candidate for therapeutic strategies in endotoxemic shock.

L4 ANSWER 13 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:295549 CAPLUS

DOCUMENT NUMBER: 120:295549

TITLE: Lipopolysaccharide (LPS)-binding protein and soluble CD14 function as accessory molecules for LPS-induced changes in endothelial barrier function, *in vitro*

AUTHOR(S): Goldblum, Simeon E.; Brann, Terrence W.; Ding, Xueda; Pugin, Jerome; Tobias, Peter S.

CORPORATE SOURCE: Div. Infect. Dis., Veterans Aff., Baltimore, MD, 21201, USA

SOURCE: Journal of Clinical Investigation (1994), 93(2), 692-702

DOCUMENT TYPE: CODEN: JCINAO; ISSN: 0021-9738

LANGUAGE: English

AB Bacterial LPS induces endothelial cell (EC) injury both *in vivo* and *in vitro*. The authors studied the effect of *Escherichia coli* O111:B4 LPS on movement of 14C-BSA across bovine pulmonary artery EC monolayers. In the presence of serum, a 6-h LPS exposure augmented ( $P < 0.001$ ) transendothelial 14C-BSA flux compared with the media control at concns.  $\leq 0.5$  ng/mL, and LPS (10 ng/mL) exposures of  $\geq 2$ -h increased ( $P < 0.005$ ) the flux. In the absence of serum, LPS concns. of up to 10  $\mu$ g/mL failed to increase 14C-BSA flux at 6 h. The addn. of 10% serum increased EC sensitivity to the LPS stimulus by  $> 10,000$ -fold. LPS (10 ng/mL, 6 h) failed to increase 14C-BSA flux at serum concns.  $< 0.5\%$ , and max. LPS-induced increments could be generated in the presence of  $\geq 2.5\%$ . LPS-binding protein (LBP) and sol. CD14 (sCD14) could each satisfy this serum requirement; either anti-LBP or anti-CD14 antibody each totally blocked ( $P < 0.00005$ ) the LPS-induced changes in endothelial barrier function. LPS-LBP had a more rapid onset than did LPS-sCD14. The LPS effect in the presence of both LBP and sCD14 exceeded the effect in the presence of either protein alone. These data suggest that LBP and sCD14 each independently functions as an accessory mol. for LPS presentation to the non-CD14-bearing endothelial surface. However, in the presence of serum both mols. are required.

L4 ANSWER 14 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:213905 CAPLUS

DOCUMENT NUMBER: 120:213905

TITLE: Lipopolysaccharide binding protein: its role and therapeutic potential in inflammation and sepsis

AUTHOR(S): Schumann, Ralf R.; Lampert, N.; Kirschning, C.; Knopf, H. P.; Hoess, A.; Herrmann, F.

CORPORATE SOURCE: Max-Delbrueck-Cent. Mol. Med., Berlin, 13122, Germany

SOURCE: Biochemical Society Transactions (1994), 22(1), 80-2

DOCUMENT TYPE: CODEN: BCSTB5; ISSN: 0300-5127

LANGUAGE: English

AB A review, with 25 refs., discussing lipopolysaccharide-binding protein, the LBP promoter, the LPS-binding site of LBP, the LPS receptor CD14, and therapeutic intervention strategies in gram-neg. sepsis.

L4 ANSWER 15 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:594644 CAPLUS

DOCUMENT NUMBER: 121:194644

TITLE: Molecular mechanisms and therapeutical intervention

AUTHOR(S) : strategies of the sepsis syndrome: Induction pattern and function of lipopolysaccharide binding protein Schumann, Ralf R.; Kirschning, C.; Lampert, N.; Knopf, H. -P.; Aberle, H.; Herrmann, F.

CORPORATE SOURCE: Max-Delbrück-Centrum für Mol. Med. (MDC), Berlin, 13122, Germany

SOURCE: International Congress Series (1993), 1042(BIOLOGY OF VITRONECT), 249-56

DOCUMENT TYPE: CODEN: EXMDA4; ISSN: 0531-5131

LANGUAGE: English

AB A review with 28 refs. Lipopolysaccharide (LPS) or endotoxin, a part of the outer membrane of gram-neg. bacteria, initiates a cascade of events in the host organism when released into the bloodstream. Moderate activation of immune cells by LPS can be beneficial, in an uncontrolled fashion, however, it often leads to severe malfunctions of the organism. Hypotension, fever, multi-organ-failure, disseminated intravascular coagulation and the full gram-neg. shock syndrome can be induced by the entry of even small amounts of LPS into the bloodstream. The sepsis syndrome has a high mortality rate and as to now no therapeutical intervention strategy has been established. With the recent discovery of binding proteins and receptors for LPS, insight in the endotoxin recognition and cell activation processes has been gained over the last years. Here the LPS binding protein LBP is discussed, focussing on its synthesis in the liver and the anal. of the promoter region of the gene. Understanding of the complex mechanism of endotoxin recognition might ultimately lead to therapeutical approaches to stop the chain reaction initiated by LPS, that leads to the shock syndrome.

L4 ANSWER 16 OF 17 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1993:515238 CAPLUS

DOCUMENT NUMBER: 119:115238

TITLE: Lipopolysaccharide binding protein and CD14 interaction induces tumor necrosis factor-.alpha. generation and neutrophil sequestration in lungs after intratracheal endotoxin

AUTHOR(S) : Ishii, Yoshiki; Wang, Yan; Haziot, Alain; Del Vecchio, Peter J.; Goyert, Sanna M.; Malik, Asrar B.

CORPORATE SOURCE: Dep. Physiol. Cell Biol., Albany Med. Coll., Albany, NY, 12208, USA

SOURCE: Circulation Research (1993), 73(1), 15-23

DOCUMENT TYPE: CODEN: CIRUAL; ISSN: 0009-7330

LANGUAGE: English

AB It has been proposed that lipopolysaccharide (LPS) bound to the 60-kD LPS binding protein (LBP) forms an LPS/LBP complex that, in turn, binds to the CD14 receptor on monocytes/macrophages and stimulates the release of cytokines. The role was examined of LBP and CD14 in tumor necrosis factor-.alpha. (TNF-.alpha.) production and neutrophil (PMN) sequestration in lungs induced by intratracheal instillation of LPS using rabbit lungs perfused at constant flow with lactated Ringer-albumin solution. LPS alone (*Salmonella minnesota*, wild type; 20 ng) or in the presence of LBP (500 ng) was injected intratracheally. In some experiments, human PMNs (5 times 10<sup>7</sup>) were added to the perfusate after a 2-h period of perfusion. Samples of lung perfusate were collected every 30 min for 180 min when bronchoalveolar lavage was also performed. TNF-.alpha. concentrations in the perfusate and bronchoalveolar lavage fluid were determined by use of a bioassay with L-929 fibroblasts, and PMN accumulation in lungs was determined by myeloperoxidase assay of lung homogenates. LPS alone did not increase TNF-.alpha. production or lung PMN accumulation, whereas the LPS/LBP complex increased TNF-.alpha. concentration in perfusate 2-fold and PMN accumulation 2-fold compared with the effect of LPS alone. Intratracheal instillation and anti-CD14 monoclonal antibody MY4 (40 μg) with the LPS/LBP complex prevented TNF-.alpha. release and PMN sequestration, whereas an isotype-matched control monoclonal antibody was ineffective. Therefore, LBP in the airspace enhances the LPS effect on TNF-.alpha. production via a CD14-dependent pathway, and as a result, CD14 activation can contribute to lung PMN sequestration. Airspace accumulation of LBP secondary to increased vascular and airway epithelial injury may play a critical role in development of acute lung injury by promoting TNF-.alpha. production via a CD14-dependent mechanism.

L4 ANSWER 17 OF 17 CAPLUS COPYRIGHT 2003 ACS  
ACCESSION NUMBER: 1994:72621 CAPLUS  
DOCUMENT NUMBER: 120:72623  
TITLE: Study of the utility of lipopolysaccharide-binding protein for diagnosis and treatment of endotoxemia  
AUTHOR(S): Ulevitch, R. J.  
CORPORATE SOURCE: Scripps Res. Inst., La Jolla, CA, USA  
SOURCE: Report (1992), Order No. AD-A259971, 12 pp. Avail.: NTIS  
From: Gov. Rep. Announce. Index (U. S.) 1993, 93(10), Abstr. No. 329,747  
DOCUMENT TYPE: Report  
LANGUAGE: English  
AB The interactions of LPS-binding protein (LBP) were defined with various isolates of bacterial endotoxin (lipopolysaccharide, LPS) or lipid A partial structures. The authors' studies include direct binding measurements and evaluation of the biol. activity of LPS-LBP complexes. These structure/function studies demonstrate the utility of LBP as a detecting reagent for an assay to measure LPS in biol. fluids. To facilitate this, the authors have also developed methods to prep. large quantities of recombinant LBP and have produced monoclonal antibodies to human LBP. All of the requisite reagents and basic information are available to facilitate development of a clin. useful assay for LPS. The major limitation in this effort is the termination of funding of this project.

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(FILE 'HOME' ENTERED AT 12:01:19 ON 08 APR 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH' ENTERED AT 12:01:40 ON 08 APR 2003

L1 55506 S SEPTICEMIA  
L2 2979 S LIPOPOLYSACCHARIDE BINDING PROTEIN  
L3 19 S L1 (P) L2  
L4 17 DUPLICATE REMOVE L3 (2 DUPLICATES REMOVED)

=> s l4 (p) treat?

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH  
FIELD CODE - 'AND' OPERATOR ASSUMED 'L28 (P) TREAT?'  
L5 3 L4 (P) TREAT?

=> d 15 1-3 ibib abs

L5 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2003 ACS  
ACCESSION NUMBER: 1998:59315 CAPLUS  
DOCUMENT NUMBER: 128:106451  
TITLE: Nonoral preparations containing phospholipids, and prevention and treatment of septicemia with them  
INVENTOR(S): Kanagi, Yoshiaki; Ono, Yoichi; Mori, Masato; Akiyama, Tadashi  
PATENT ASSIGNEE(S): Nippon Seiyaku K. K., Japan  
SOURCE: Jpn. Kokai Tokkyo Koho, 8 pp.  
CODEN: JKXXAF  
DOCUMENT TYPE: Patent  
LANGUAGE: Japanese  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 10017476	A2	19980120	JP 1996-188173	19960628

PRIORITY APPLN. INFO.: JP 1996-188173 19960628

AB The preps. for \*\*\*septicemia\*\*\* contain phospholipids suspended therein, and the phospholipids mobilize lipoproteins when i.v. administered, thus inactivating endotoxins and/or endotoxin- \*\*\*lipopolysaccharide\*\*\* - \*\*\*binding\*\*\* \*\*\*protein\*\*\* complexes. \*\*\*Septicemia\*\*\* is prevented or \*\*\*treated\*\*\* by i.v. administration of the preps. Thus, yolk lecithin was homogenized with an aq. glycerin soln. to give a suspension. Escherichia coli LPS was injected to rats on 4th day of 5-day consecutive i.v. administration of

the suspension to cause no death of the animals.

L5 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1995:982430 CAPLUS

DOCUMENT NUMBER: 124:764

TITLE: Recombinant preparation of polypeptides of human lipopolysaccharide binding protein and use of the polypeptides for sepsis treatment

INVENTOR(S): Han, Jiahui; Ulevitch, Richard J.; Tobias, Peter S.

PATENT ASSIGNEE(S): Scripps Research Institute, USA

SOURCE: PCT Int. Appl., 63 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9525117	A1	19950921	WO 1995-US3384	19950315
W: AU, CA, FI, JP, NO				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5837810	A	19981117	US 1994-215089	19940315
AU 9521868	A1	19951003	AU 1995-21868	19950315
PRIORITY APPLN. INFO.:			US 1994-215089	19940315
			WO 1995-US3384	19950315

AB A polypeptide fragment of lipopolysaccharide (LPS)-binding protein (LBP) that inhibit the binding of LPS released by gram-neg. bacteria into the CD14 receptor is provided. A method of ameliorating symptoms of sepsis in a subject by administration of an LBP polypeptide of the invention, or administration of antibody to LBP polypeptide, or anti-idiotype antibody is also disclosed.

L5 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:72623 CAPLUS

DOCUMENT NUMBER: 120:72623

TITLE: Study of the utility of lipopolysaccharide-binding protein for diagnosis and treatment of endotoxemia

AUTHOR(S): Ulevitch, R. J.

CORPORATE SOURCE: Scripps Res. Inst., La Jolla, CA, USA

SOURCE: Report (1992), Order No. AD-A259971, 12 pp. Avail.: NTIS

From: Gov. Rep. Announce. Index (U. S.) 1993, 93(10), Abstr. No. 329,747

DOCUMENT TYPE: Report

LANGUAGE: English

AB The interactions of LPS-binding protein (LBP) were defined with various isolates of bacterial endotoxin (lipopolysaccharide, LPS) or lipid A partial structures. The authors' studies include direct binding measurements and evaluation of the biol. activity of LPS-LBP complexes. These structure/function studies demonstrate the utility of LBP as a detecting reagent for an assay to measure LPS in biol. fluids. To facilitate this, the authors have also developed methods to prep. large quantities of recombinant LBP and have produced monoclonal antibodies to human LBP. All of the requisite reagents and basic information are available to facilitate development of a clin. useful assay for LPS. The major limitation in this effort is the termination of funding of this project.

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L3 19 S L1 (P) L2

L4 17 DUPLICATE REMOVE L3 (2 DUPLICATES REMOVED)

L5 3 S L4 (P) TREAT?

=> s bactericidal/permeability increasing protein  
MISSING OPERATOR  
MISSING OPERATOR  
MISSING OPERATOR  
MISSING OPERATOR  
MISSING OPERATOR

=> s bactericidal/permeability (w) increasing (w) protein  
'PERMEABILITY' IS NOT A VALID FIELD CODE  
L6 0 BACTERICIDAL/PERMEABILITY (W) INCREASING (W) PROTEIN

=> s bactericidal (w) permeability (w) increasing (w) protein  
L7 2136 BACTERICIDAL (W) PERMEABILITY (W) INCREASING (W) PROTEIN

=> s (limulus anti-lipopolysaccharide factor) or lalf  
L8 72 (LIMULUS ANTI-LIPOPOLYSACCHARIDE FACTOR) OR LALF

=> s l2 (p) (17 or 18)  
L9 317 L2 (P) (L7 OR L8)

=> s l9 (p) hybrid  
L10 6 L9 (P) HYBRID

=> duplicate remove l10  
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'  
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n  
PROCESSING COMPLETED FOR L10  
L11 2 DUPLICATE REMOVE L10 (4 DUPLICATES REMOVED)

=> d l11 1-2 ibib abs

L11 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS  
ACCESSION NUMBER: 1995:492022 CAPLUS  
DOCUMENT NUMBER: 122:232671  
TITLE: Lipopolysaccharide binding protein derivatives, their manufacture with recombinant cells, and their use in treatment of Gram-neg. bacterial infections  
INVENTOR(S): Gazzano-Santoro, Helene; Theofan, Georgia; Trown, Patrick W.  
PATENT ASSIGNEE(S): Xoma Corp., USA  
SOURCE: PCT Int. Appl., 108 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 2  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9500641	A1	19950105	WO 1994-US6931	19940617
W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, KZ, LK, LU, LV, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SK, UA, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9471756	A1	19950117	AU 1994-71756	19940617
PRIORITY APPLN. INFO.:			US 1993-79510	A 19930617
			WO 1994-US6931	W 19940617
AB Disclosed are novel biol. active ***lipopolysaccharide*** ***binding*** ***protein*** (LBP) derivs. including LBP deriv. ***hybrid*** proteins which are characterized by the ability to bind to and neutralize LPS and which lack the CD14-mediated immunostimulatory properties of holo-LBP. CDNA's for human LBP and for (1-197)LBP, called LBP25 were cloned. Genes for LBP25, for BPI23 [where BPI refers to human ***bactericidal*** / ***permeability*** - ***increasing*** ***protein*** and BPI23 to (1-199)BPI], and ***hybrid*** LBP-BPI proteins were constructed and expressed in CHO cells. Lipid A binding activity and pharmacokinetics of selected proteins were examd. LBP25,				

unlike LBP, did not potentiate release of tumor necrosis factor by peripheral blood mononuclear cells and did not mediate LPS-stimulated tissue factor prodn. LBP25 completely inhibited LPS induction of endothelial cell adhesiveness for neutrophils. Addnl., LBP25 was unable to mediate CD14-dependent enhanced binding of bacteria to monocytes.

L11 ANSWER 2 OF 2 MEDLINE DUPLICATE 1  
ACCESSION NUMBER: 94179366 MEDLINE  
DOCUMENT NUMBER: 94179366 PubMed ID: 8132678  
TITLE: Complete cDNA encoding human phospholipid transfer protein from human endothelial cells.  
AUTHOR: Day J R; Albers J J; Lofton-Day C E; Gilbert T L; Ching A F; Grant F J; O'Hara P J; Marcovina S M; Adolphson J L  
CORPORATE SOURCE: Department of Medicine, University of Washington, Seattle 98103.  
CONTRACT NUMBER: HL 30086 (NHLBI)  
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Mar 25) 269 (12) 9388-91.  
Journal code: 2985121R. ISSN: 0021-9258.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
OTHER SOURCE: GENBANK-L26232  
ENTRY MONTH: 199404  
ENTRY DATE: Entered STN: 19940428  
Last Updated on STN: 19940428  
Entered Medline: 19940421

AB Phospholipid transfer protein, with an apparent molecular mass of 81 kDa, was purified from human plasma. The NH<sub>2</sub>-terminal amino acid sequence of a 51-kDa proteolytic fragment obtained from phospholipid transfer protein allowed degenerate primers to be designed for polymerase chain reaction and the eventual isolation of a full-length cDNA from a human endothelial cDNA library. The cDNA is 1,750 base pairs in length and contains an open reading frame of 1,518 nucleotides encoding a leader of 17 amino acids and a mature protein of 476 residues. Northern blot analysis shows a single mRNA transcript of approximately 1.8 kilobases with a wide tissue distribution. The gene was mapped to chromosome 20 using a human/rodent somatic cell \*\*\*hybrid\*\*\* mapping panel. Phospholipid transfer protein was found to be homologous to human cholesteryl ester transfer protein, human \*\*\*lipopolysaccharide\*\*\* - \*\*\*binding\*\*\* \*\*\*protein\*\*\*, and human neutrophil \*\*\*bactericidal\*\*\* \*\*\*permeability\*\*\* \*\*\*increasing\*\*\* \*\*\*protein\*\*\* (20, 24, and 26% identity, respectively).

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L5 3 S L4 (P) TREAT?  
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L7 2136 S BACTERICIDAL (W) PERMEABILITY (W) INCREASING (W) PROTEIN  
L8 72 S (LIMULUS ANTI-LIPOPOLYSACCHARIDE FACTOR) OR LALF  
L9 317 S L2 (P) (L7 OR L8)  
L10 6 S L9 (P) HYBRID  
L11 2 DUPLICATE REMOVE L10 (4 DUPLICATES REMOVED)

=> s 13 (p) mutant

L12 0 L3 (P) MUTANT

=> d his

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L6 0 S BACTERICIDAL/PERMEABILITY (W) INCREASING (W) PROTEIN  
L7 2136 S BACTERICIDAL (W) PERMEABILITY (W) INCREASING (W) PROTEIN  
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L9 317 S L2 (P) (L7 OR L8)  
L10 6 S L9 (P) HYBRID  
L11 2 DUPLICATE REMOVE L10 (4 DUPLICATES REMOVED)  
L12 0 S L3 (P) MUTANT

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	96.72	96.93

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-11.72	-11.72

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